

Dental Caries*

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Since the beginning of this century it has been widely recognized that micro-organisms constitute an essential factor in the pathogenesis of dental caries. Indirect proof of this concept came when McClure and Hewitt (1946) observed that the inclusion of penicillin in a normally cariogenic diet resulted in a substantial reduction of caries in rats. Using gnotobiotic techniques, Orland *et al.* (1954) showed that rats fed a diet which is normally cariogenic failed to develop dental decay in the absence of bacteria. This observation was confirmed and extended by Fitzgerald and Keyes (1960).

It was Keyes (1960), however, who showed that caries is an infectious and transmissible disease

in the hamster (Fig. 1). A litter of hamsters taken from a strain which had been caries-active for 4 generations was divided into 2 groups. One-half was given antibiotics in its feed to suppress the cariogenic flora; the other half constituted the control. The antibiotic-fed group remained caries free, while other animals developed rampant decay. A litter was obtained from a female in each group. That from the caries-inactive animal was divided into two; half the litter was housed alone and the other half with the litter from the caries-active animals. The half-litter housed alone remained caries free whereas the remaining animals developed rampant caries.

The significance of these experiments for humans has become increasingly clear. Zinner *et al.* (1965) observed that rodents infected with specific streptococci isolated from carious lesions in humans developed rampant dental caries. This work has been confirmed and extended by Krasse (1966), Gibbons *et al.* (1966) using rodents, and by Bowen (1969a) using monkeys as experimental animals (Fig. 2). These observations may be of more significance for humans than would first appear. After examination of more than 1000 families, Klein (1946) found a clear correlation between the caries score of sibs and that of their parents. Böök and Grahnen (1953), too, have observed that parents and sibs of caries-free adults had significantly less caries than those of the caries-active control adults. The prevalence of caries in sons and daughters was compared with that in mothers and fathers by Davies (1965) in a population living in a remote Pacific Island. He found a highly significant association between the caries scores of mothers and sons and between those of mothers and daughters, but none between those of fathers and their children. A nonbacteriological explanation could be offered for these observations; however it has been shown that there is a positive correlation between the numbers of *Streptococcus mutans* and the degree of caries activity in humans (de Stoppe-laar, van Houste, and Backer Dirks, 1969) and also in animals (Krasse, 1966; Bowen, 1969a). In addition it has been observed that the bacterial

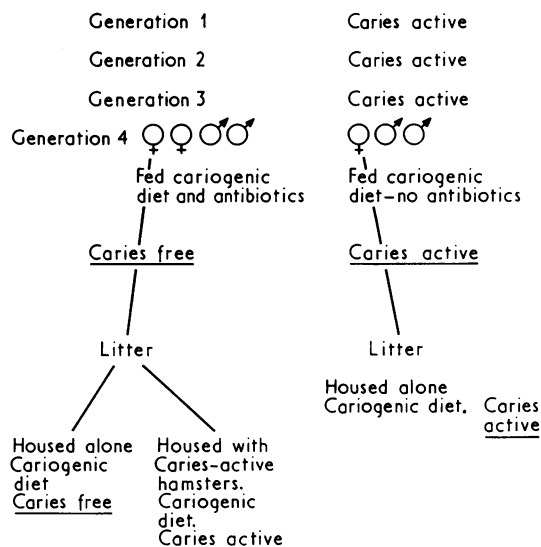


FIG. 1.—The effect of depressing the maternal 'cariogenic flora' on the incidence of dental caries in offspring (after Keyes, 1960).

*Few paediatricians, we believe, are in touch with the current views of their dental colleagues on the subject of dental caries and their prevention, and we therefore invited the author of this article to review the subject.—Editors.

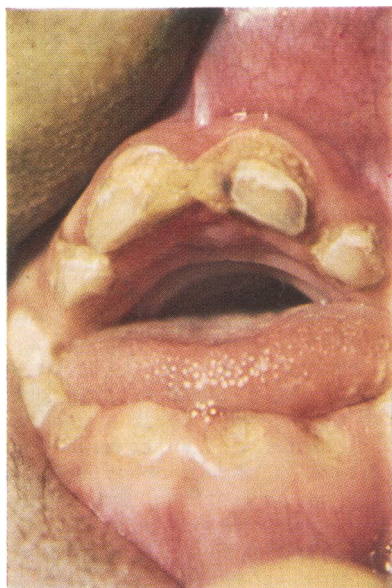


FIG. 2.—Rampant dental caries in a/a teeth of a young monkey infected with *Strep. mutans* and fed a diet rich in carbohydrate.

population of the mouth declines markedly after the restoration of carious lesions (Shklair *et al.*, 1956). It seems probable, therefore, that the mother with active carious lesions is more likely to infect her child than the mother who is free of caries.

These cariogenic micro-organisms which are grouped as *Strep. mutans* (Clarke, 1924) have many unique characteristics. It appears that they can only inhabit the mouth if there is a solid surface for them to colonize (Carlsson, Soderholm, and Almfeldt, 1969; Cornick and Bowen, 1971), and because of this it has been suggested that they be termed 'obligate periphytes'. After a thorough cleaning of enamel, *Strep. mutans* are among the first to recolonize the tooth surface (van Houte, Gibbons, and Pulkkinen, 1971).

Strep. mutans also has the capacity to form extracellular polysaccharides, such as dextrans and levans, from the wide range of carbohydrates. These substances contribute to the adhesiveness of the micro-organisms and add significantly to the bulk of the soft white tenacious material (plaque) found on most tooth surfaces. The precise role of these materials in plaque is uncertain. They can protect the micro-organisms against inimical influences, act as reserves of carbohydrate, and prevent neutralizing substances diffusing through the plaque.

These bacteria also have the capacity to form acid

rapidly from a wide variety of common sugars.

Though micro-organisms are essential in the pathogenesis of dental caries, their mere physical presence on the tooth surface will not of itself give rise to dental decay. Diet, too, plays an essential role in the development of this disease. This was shown by Kite, Shaw, and Sognnaes (1950), who observed that rodents that received their diet by stomach tube remained caries free. Though plaque forms in the absence of food, it lacks the capacity to form acid when provided with a solution of sucrose (Bowen and Cornick, 1970). This is only one illustration of the capacity of plaque to form acid being influenced by the composition of the diet. Diets rich in carbohydrate result in a plaque with an enhanced acidogenic capacity (Bowen and Cornick, 1967). Though the total content of carbohydrate in a diet influences the acid-producing capacity of plaque it appears that it is the frequency with which it is ingested that determines its ultimate cariogenicity. There is a strong positive correlation between the frequency of ingestion and the incidence of dental caries (Gustafsson *et al.*, 1954). Each specific intake of fermentable carbohydrate is followed by a rapid production of acid; frequently pH values as low as 4 occur after 20 minutes, resulting in the dissolution of enamel. An extreme example of the consequence of this can be seen in so-called comforter caries, where the upper incisors and palatal surfaces of molars decay rapidly in young children who suck on dinky feeders filled with sugar solution (James, Parfitt, and Falkner, 1957; Winter, Hamilton, and James, 1966).

There is, however, little correlation between the total amount of sugar consumed and caries experience (Zita, McDonald, and Andrews, 1959).

The microbial population of the plaque can be influenced by the composition of the diet. Bowen and Cornick (1967) have observed that the population of organisms forming extracellular polysaccharides declined markedly after restriction in the carbohydrate intake of monkeys; more recently de Stoppelaar, van Houte, and Backer Dirks (1970) have made similar observations in humans. Available evidence (Krasse *et al.*, 1967; Cornick and Bowen, 1971) also suggests that sucrose may aid the implantation of *Strep. mutans* into mouths of humans and some animals. Though the pathogenicity of plaque is influenced by its microbial population and also by dietary composition, the period for which plaque is allowed to accumulate will also influence its capacity to produce acid and its response to buffers such as saliva. The older the plaque the greater the fall in pH after the application

of sugar. Furthermore, it is more difficult to neutralize acid formed in old plaque than that in young plaque (Graf and Muhlemann, 1966; D. E. R. Cornick, and W. H. Bowen, unpublished).

It is frequently argued that sucrose is the 'arch criminal' (Newbrun, 1969) in the aetiology of dental caries and that all other sugars are innocent in this respect. This concept has probably arisen in part at least from the observation that patients suffering from fructose intolerance, and therefore unable to consume foods containing fructose or sucrose, have a low prevalence of dental caries. It is clear that those people must modify their dietary habits in many ways other than merely avoiding sucrose-containing foods. Sucrose is also the sugar most frequently consumed by humans (55 kg per head annually in U.K.). It accounts for more than 30% of all carbohydrate intake (Yudkin, 1964). As there is a positive correlation between the frequency of intake of carbohydrate and the incidence of caries, it is not surprising to find sucrose condemned. However, this unfortunately has been extrapolated to the unfounded and dangerous assumption that a significant reduction in caries prevalence would occur if sucrose were replaced by other sugars. There is an abundance of evidence (Campbell and Zinner, 1970; Green and Hartles, 1969; Shaw, Krumins, and Gibbons, 1967) that shows that animals fed diets composed largely of simple sugars other than sucrose develop carious lesions.

The need for effective prevention is great. The toll of pain and suffering cannot be measured in simple economic terms; it has, however, been estimated that approximately 1.75 million working days are lost each year through dental disease. In addition, approximately 70 million pounds is spent annually in the National Health Service repairing carious teeth or their consequences.

Measures for the prevention of dental decay can be directed towards strengthening teeth against carious attack, interfering with the metabolism of bacteria, and the alteration of eating habits.

The most effective methods are likely to be those that do not rely on the individual for their application. In addition it is unlikely in present circumstances that the dentist can make a really effective contribution to prevention on a public health basis because the majority of his time must of necessity be devoted to treatment. Furthermore, fewer than 30% of the population attend a dentist regularly. By far the most effective method of prevention is the addition of fluoride (1 ppmF) to drinking water. This method has been acclaimed as being safe and effective by medical and dental associations

throughout the world. It has been endorsed by the World Health Organization.

The proportion of children free from dental decay has risen dramatically in certain areas of the United Kingdom (Ministry of Health, 1969; Beal and James, 1971) after the introduction of water fluoridation, and increased from 15 to 40% in 3- to 7-year-old children over a period of 11 years. Water fluoridation exerts its greatest effect on the free smooth surfaces (buccal and lingual); approximately 80 to 90% reduction in incidence is achieved. Much of this reduction may be due to a posteruptive topical effect, traditionally thought to be locally mediated but, in fact, attributable to systemic administration as well (W. H. Bowen, unpublished). The protective effect is less in pits and fissures than on other surfaces and little prolonged protection is achieved in these sites unless teeth have been exposed to fluoride from the earliest stages of development.

The application of topical fluoride in such preparations as toothpaste and mouthwashes leads to a small but significant reduction in the incidence of caries if conscientiously applied.

Recently attention has been focused on the use of plastics such as cyanoacrylates to seal off fissures as a means of preventing dental decay in pits and fissures which are the most vulnerable surfaces. This idea represents the resurrection of an old concept described by Hyatt (1936). He suggested the cutting of shallow cavities in such surfaces before they became carious and filling them with amalgam. The procedure was described as 'prophylactic odontotomy' and was based on the belief that as the vast majority of occlusal pits and fissures decay within 2 years of eruption it is better to fill these teeth before decay could advance and destroy a large amount of tooth surface.

The development of new plastic materials has led to a more sophisticated approach. The sites to be treated are thoroughly cleaned, etched briefly with phosphoric acid, rinsed, and dried. The plastic material is then applied. A substantial degree of protection has been achieved by some investigators (Buoncore, 1970), while others have found it to be a complete failure (Parkhouse and Winter, 1971). There is little doubt, however, that new materials will be developed which will be more effective than those in current use.

The outstanding advantages of this procedure are that it is painless and does not require cutting of the tooth. It does however require meticulous attention to detail and would require a great deal of manpower to administer on a mass scale; the cost may exceed that of a conventional filling. Ideally,

too, teeth should be treated soon after eruption, and as the most susceptible teeth do not all erupt together, it would require many visits to the dental surgery to treat all high-risk teeth. It does not of course protect the smooth surfaces. It could well be that the protection of one surface, albeit the most susceptible, is a futile exercise if other surfaces are ignored. The occlusal surfaces have to be cut in most instances, if caries develops on the interproximal surfaces.

Reduction in the frequency of intake of fermentable carbohydrate is often advocated as a means of preventing caries. Though it is true that this does lead to a significant reduction of caries incidence, it calls for so much self discipline that it is not really practical on a community basis. The pleasure of consuming sweets, biscuits, or carbonated beverages is not related by the patient to the need for dental treatment at some future time.

The use of phosphates such as trimetaphosphate as a possible means of preventing dental decay has attracted a great deal of attention (Harris, 1970). Many of these substances have been effective in rodents but have not been successful when tested in humans, probably because of the difficulty of maintaining contact with the tooth surface of sufficient duration. Recently (Bowen, 1972) calcium glycerophosphate has been shown to reduce the incidence of dental caries in monkeys when fed at a concentration of 1% in some components of their diet. Trials in humans are in progress.

Enzymes such as dextranase are effective in reducing dental decay in hamsters (Fitzgerald *et al.*, 1968) and also in monkeys (Bowen, 1971). The results of short-term trials carried out in humans assessing the effect of dextranase-containing mouthwashes on plaque have been equivocal (Caldwell *et al.*, 1970; Keyes *et al.*, 1970; Lobene and Soparkar, 1970). The difficulty of obtaining prolonged contact with plaque, coupled with the heat instability of the enzyme, seems to preclude the possibility of using dextranase in its present form as a method for preventing dental caries. However, research is in progress to determine whether substances can be developed which would lead to a binding of dextranase or similar enzymes to plaque resulting in prolonged activity.

With the identification of specific bacteria capable of inducing rampant dental caries in animals, the possibility of developing a vaccine became an attractive possibility. Such a prospect may seem remote particularly by those who have come to regard rotting teeth as normal. In recent years a small group of monkeys has been successfully

vaccinated against dental caries (Bowen, 1969b) using whole live streptococci. Wagner (1966) and Bahn *et al.* (1969) have made similar observations using rats as experimental animals. Support for the concept of vaccine against dental caries is found in work of Lehner, Cardwell, and Clarry (1967) who observed that caries-free subjects have higher levels of immunoglobulins in their saliva than caries-active subjects. An inverse relation has been reported by Zengo *et al.* (1971) between the concentration immunoglobulin IgA submandibular saliva and the level of caries activity in humans. Though IgA is the predominant immunoglobulin present in saliva, small amounts of IgG are also present. It is frequently argued that the concentration of immunoglobulins in saliva is too low to have any significant protective effect. However, it should be remembered that approximately 1200 ml saliva is secreted per day; clearly, therefore, the total amount of immunoglobulin that appears in the mouth could amount to as much as 250 mg or more per day.

It is clear that whole bacterial cells would not be acceptable as a suitable vaccine if only because these micro-organisms have been implicated in the pathogenesis of bacterial endocarditis. Research is continuing into determining the antigenicity of such substances as dextran-sucrases, which synthesize dextrans from sucrose, and components of cell walls. The immunoglobulin response using different sites of vaccination is also being investigated. Clearly much remains to be done before a vaccine which is both safe and effective will be available.

Our understanding of the aetiology of dental caries has increased greatly in recent years. It is clear that the disease is not simply a case of 'too many sweets'. Sufficient is known to indicate that if people were to alter their social habits significantly, dental caries could to a large extent be controlled. However, it is unlikely that any disease which affects more than 95% of the population will be controlled to any significant degree by individual effort. Effective prevention calls for public health measures, and of these water fluoridation is the most effective and the simplest to administer.

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